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LETTER TO THE EDITOR

Chlamydophila pneumoniae infection as a trigger for Vogt-Koyanagi-Harada syndrome

Infección por *Chlamydophila pneumoniae* como desencadenante del síndrome de Vogt-Koyanagi-Harada

Dear Editor:

The Vogt-Koyanagi-Harada syndrome (VKHS) is a systemic granulomatous autoimmune disease, targeting melanocytic self-antigens. Its main symptoms include ocular, neurological, auricular, and integumentary manifestations. The condition presents 4 stages: the prodromal, acute uveitic, convalescent, and chronic recurrent phases. The prodromal stage usually manifests with headache and meningism, with ocular symptoms appearing later.[1,2] Considering the relevance of establishing early immunosuppressive treatment to avoid recurrence and prevent eye involvement, it is essential to maintain a high level of clinical suspicion and to establish a correct diagnosis.

We present the case of a 58-year-old Moldavian woman with history of major depressive disorder and dyslipidaemia without active treatment. She attended the emergency department 3 times due to acute-onset headache, photopsias, and blurred vision. She presented arterial hypertension at all visits. An eye fundus examination revealed bilateral optic disc oedema and the patient was admitted to the neurology department due to suspicion of intracranial hypertension. During admission, a brain MRI study showed bilateral choroidal effusion but no parenchymal lesions (Fig. 1).

Considering the MRI findings, a lumbar puncture was performed, revealing an opening pressure of 20.5 cm H_2O. The CSF analysis showed 320 leukocytes/mm^3 (predominantly lymphocytes), 10 erythrocytes/mm^3, a CSF/plasma glucose ratio of 0.49, and protein level of 1.03 g/L. Cultures and PCR results for neurotropic viruses were negative, as were cytology findings. In the light of these results, we performed an ophthalmological evaluation with optical coherence tomography (OCT) and fluorescein angiography, revealing bilateral panuveitis with choroidal effusion and serous retinal detachment.

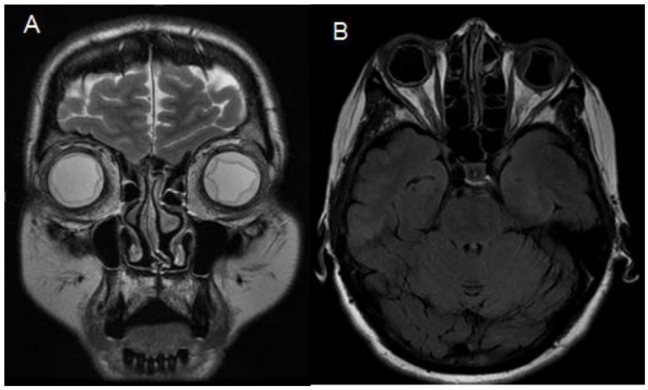

Figure 1 Coronal T2-weighted (A) and axial T2-FLAIR (B) MRI sequences revealing bilateral choroidal effusion with non-haemorrhagic exudates and bilateral papilloedema.

Overall, the patient's symptoms and test findings suggest uveo-meningeal syndrome, with the patient presenting aseptic meningitis, which together with the ophthalmological findings indicates VKHS.

The study was expanded with serology tests for Epstein-Barr virus, cytomegalovirus, herpes simplex virus, measles, rubella, mumps, *Toxoplasma gondii*, hepatitis B virus, hepatitis C virus, *Coxiella*, *Rickettsia*, *Brucella*, *Mycoplasma*, HIV, and *Treponema pallidum*; all findings were negative for active or recent infection. The only exception was *Chlamydophila pneumoniae*, which showed high IgG titres, suggesting recent infection.

Thus, the patient met diagnostic criteria for incomplete VKHS.[3] Treatment was immediately started with intravenous methylprednisolone dosed at 1000 mg/24 h for 5 days, followed by oral prednisone at 1 mg/kg/day for a month and a half, as tapering was necessary due to adverse drug reactions. She also received topical treatment with cycloplegics and dexamethasone. The patient improved considerably after treatment onset, with visual acuity increasing from 0.1 to 0.4 bilaterally at 6 days of treatment and 1/1 at 4 months of treatment. As the patient did not tolerate high-dose corticosteroids, and with a view to better controlling inflammation and choroidal neovascularisation, we added azathioprine to the treatment schedule.[4—6] Treatment response was also monitored using OCT, which revealed a fast, remarkable resolution of retinal detachment and choroidal inflammation.

The initial treatment of this syndrome is currently a subject of debate. The classical approach consists of initial treatment with corticosteroids, followed by immunotherapy at later stages. Over the last decade, we have observed a

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clear correlation between early onset and high-dose intravenous glucocorticoids (between 500 and 1000 mg for 3 days), and initial clinical improvement.[7] Furthermore, some recent studies support the use of concomitant therapy with immunosuppressants from onset, as data suggest that this combination therapy is associated with a decrease in the number of late complications.[8] However, this is not implemented in everyday clinical practice.

In the case of our patient, we initially used intravenous monotherapy at 1000 mg for 5 days, (longer than the classical exposure time to high-dose immunosuppression), obtaining very good clinical outcomes with resolution of neurological symptoms and the choroidal inflammation shown by OCT.

We should underscore the recent *C. pneumoniae* infection as a possible trigger of the disease. VKHS is known to be triggered by various (mainly viral) infections, however, we found no cases in the literature that were triggered by *C. pneumoniae* infection, which has been associated with such other autoimmune diseases as Kawasaki disease or multiple sclerosis.[9]

Conflicts of interest

The authors have no conflicts of interest to declare.

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Case report: is there a link between morning glory syndrome and TANGO2 mutation disease, or is this an unusual association?

¿Existe relación entre el síndrome de *morning glory* y la enfermedad por mutación en el gen TANGO2, o es una asociación inusual?

Introduction

Congenital anomalies of the optic nerve are a rare but significant cause of visual deficits.[1] In 1970, Peter Kindler described for the first time a sporadic congenital anomaly of the optic nerve whose cause was not fully understood; involvement was predominantly unilateral, and characterised by an abnormally large optic disc, large excavation, presence of glial tissue remnants, and a radial peripapillary microvascular network. He named the condition morning glory syndrome. Most of these cases manifest during childhood or adolescence, predominantly affecting girls, presenting with symptoms of visual deficit and strabismus.[2]

Case report

Our patient was a 32-year-old woman, born to first-degree consanguineous parents and with history of psychomotor retardation, moderate intellectual disability, non-autoimmune hypothyroidism, and bilateral sensorineural hearing loss. She was being followed up by the neurology department due to episodes of disorientation.

The examination revealed generalised hyperreflexia with increased reflexogenic zone, absence of clonus, and bilateral positive Hoffman sign. The ophthalmological examination revealed exotropia of the right eye. Direct ophthalmoscopy showed right optic disc pallor, and indirect ophthalmoscopy revealed increased size and excavation of the right optic disc. The papilla was surrounded by an abnormally pigmented chorioretinal ring with marked peripapillary atrophy and a radial layout of blood vessels. The centre of the optic disc was covered by whitish glial tissue remnants. The peripheral retina and fovea of the right eye were normal. An A- and B-mode ultrasound study ruled out serous retinal detachment,

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